

Incidence of conduction defects in African and Coloured patients with congestive cardiomyopathy

Elliot Chesler and Walter Beck

From the Cardiac Clinic, Groote Schuur Hospital, and the Department of Medicine, University of Cape Town, Cape Town, South Africa

The electrocardiograms of 195 patients (78 Coloured and 117 Bantu) presenting with congestive cardiomyopathy have been studied. The tracings had a fairly uniform pattern showing left ventricular hypertrophy in every case, frequent left atrial enlargement, the absence of right ventricular hypertrophy, and the rarity of right axis deviation in the absence of pulmonary embolism. A study of the conduction defects showed the absence of right bundle-branch block or the combination of left axis deviation and right bundle-branch block. Conduction defects involving the left bundle-branch of the conduction system were found in 25 per cent of all cases, with left bundle-branch and left anterior hemiblock occurring with equal frequency. These findings are related to fibrosis of the divisions of the left bundle-branch. A comparison of the incidence of these conduction defects showed that they were almost twice as common in the Coloured as compared to the Bantu, indicating more severe left ventricular damage. Recent evidence relating to aetiology of congestive cardiomyopathy in the Bantu is reviewed and the causative role of alcohol discussed. It is suggested that the prevalence of chronic alcoholism among the Coloured population may be responsible for the differences in the comparative incidence of the conduction defects reported here.

Among the various groups of South Africa congestive cardiomyopathy is rare in the White population whereas in the Bantu it constitutes the leading form of heart disease and has been extensively studied (Gillanders, 1951; Becker, Chatgidakis, and Van Lingen, 1953). A clinically similar condition in the Cape Coloured has not received the same attention (Schrire, 1964). One report indicated that left axis deviation was a frequent finding in the Bantu with congestive cardiomyopathy but the electrocardiogram of Coloured patients presenting with this disease has not been studied (Schamroth and Blumsohn, 1961).

We report findings of a retrospective analysis of the comparative incidence of the electrocardiographic conduction defects in the Bantu and Coloured patients who presented with congestive cardiomyopathy.

Patients and methods

In the Cardiac Clinic, Groote Schuur Hospital, there are records of 197 patients who presented in congestive cardiac failure in the absence of hypertension, valvular heart disease, beri-beri or acute myocarditis. There were two patients (ages 15 and 23) whose electrocardiograms

Received 15 January 1973.

showed abnormal Q waves suggestive of myocardial necrosis; they were excluded from the study because it was uncertain whether they were examples of myocarditis or congestive cardiomyopathy complicated by coronary embolism. The remaining 195 patients form the basis of this study and the data relating to their race, age, and sex are set out in Table 1. Characteristically, they were in severe heart failure with raised systemic venous pressure and hepatomegaly; the murmurs of functional tricuspid, or more commonly mitral insufficiency, were frequently audible. Chest x-rays confirmed the presence of pronounced cardiomegaly and pulmonary venous congestion.

Cardiac catheterization and angiography in 45 patients confirmed the presence of severe heart failure with low cardiac output, and the absence of valvular abnormality other than that of mild functional mitral and tricuspid insufficiency. Left ventricular angiograms always showed abnormalities of wall movement (akinesis, hypokinesis, and asynergy). In several instances filling defects at the apex of the left ventricle were suggestive of mural thrombus. Aortograms, particularly because of slow dye clearance, allowed clear visualization of dilated coronary arteries characteristically found in myopathic ventricles. In 19 cases, selective coronary angiography demonstrated widely patent vessels. In 14 cases, the apex of the right ventricle was biopsied using a Konno endomyocardial biotome. Light-microscopical examination of these specimens showed hypertrophied muscle fibres, but no

TABLE I Age, race, and sex distribution in 195 cases of congestive cardiomyopathy

	Coloured		Bantu	
	Men	Women	Men	Women
No. and %	41 (52.5%)	22 (28.2%) + 15 (19.2%)	80 (68.4%)	27 (23%) + 10 (8.5%)
Age (mean)	42	39	43	39
Total		78		117

Note: Among the women, the numbers following the plus signs indicate the cases of puerperal cardiomyopathy.

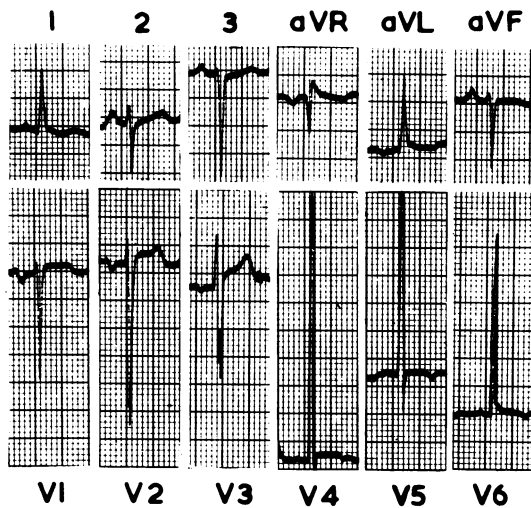


FIG. 1 Electrocardiogram showing sinus rhythm, left anterior hemiblock, left ventricular hypertrophy, and left atrial enlargement.

evidence of myocarditis or amyloid infiltration. Necropsies in 7 patients indicated findings characteristic of congestive cardiomyopathy, to be described in more detail later.

Twelve-lead electrocardiograms were available in all patients and in most instances serial tracings were obtained. The following electrocardiographic criteria were used. *Left anterior hemiblock*: mean QRS axis directed to the left at -30° or superior (Fig. 1). *Complete right bundle-branch block*: QRS duration of 0.12 sec or more with a prominent S wave in standard lead I and RSR' in V1. *Left bundle-branch block*: QRS duration of 0.12 sec or more with QS, or rS deflections in lead V1 with raised ST segments, and a wide, often notched, R pattern in V6 with depressed ST segments with absence of significant q waves in standard lead I, aVL, and V6. When the mean QRS axis was located at -30° or superior, these cases were classified as left bundle-branch block with left axis deviation (Fig. 2). *Left ventricular hypertrophy*: the sum of the R in V5 or V6 and the S in V1 exceeded 35 mm. *Left atrial enlargement*: terminal negativity of the P wave of 1 mm or more in depth and duration of 0.04 sec or more in lead V1. *Right ventricular hypertrophy*: a reversal ratio of R/S in V1 to V3 ($R/S > 1$).

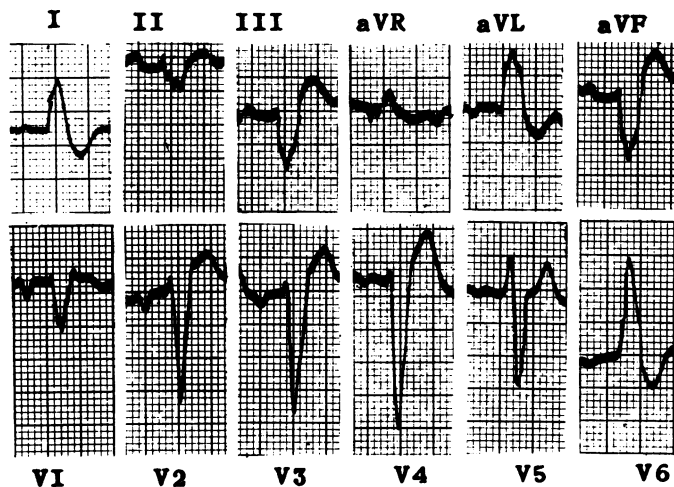


FIG. 2 Electrocardiogram showing sinus rhythm, left bundle-branch block, left axis deviation, and left atrial enlargement.

In all cases the mean QRS axis was plotted to the nearest 10 degrees on the hexaxial reference system.

Results

The mean age for both Coloured and Bantu patients presenting with congestive cardiomyopathy was similar. Among the female patients the onset of heart failure was clearly related to pregnancy in 19 per cent of the Coloured and 8.5 per cent of the Bantu patients, respectively. When these cases are excluded it will be seen from Table 1 that idiopathic congestive cardiomyopathy is much commoner among men than women (1.9/1 for Coloured and 2.9/1 for Bantu).

The majority were in sinus rhythm: the incidence of atrial fibrillation was 3.6 per cent. All 195 patients had left ventricular hypertrophy by voltage criteria and the incidence of left atrial enlargement was 88 per cent. Right ventricular hypertrophy, by the criteria specified, was not observed in any of the tracings.

The mean QRS axis, including those cases who had left bundle-branch block, was as follows. -30° to -120° : 30 cases (15%); 0° to -30° : 37 cases (19%); $+30^{\circ}$ to $+90^{\circ}$: 117 cases (60%); $+90^{\circ}$ to $+120^{\circ}$: 7 cases (3.6%); and indeterminate: 4 cases (2%).

The incidence of the various conduction defects is shown in Table 2. Left anterior hemiblock and left bundle-branch block occurred with equal frequency – 12.8 and 12.3 per cent, respectively. Right bundle-branch block alone, or in combination with left anterior hemiblock was never observed. Of the 7 cases with right axis deviation, this axis shift was temporary in 1 (Fig. 3) and in another case was related to pulmonary embolism proven at necropsy. In 10 cases (5%) the PR interval was prolonged and 3 of these were associated with left anterior hemiblock but there was no association with left bundle-branch block. Because of the absence of right bundle-branch block and second or third-degree AV block in the series it is doubtful whether these long PR intervals were a manifestation of bilateral bundle-branch

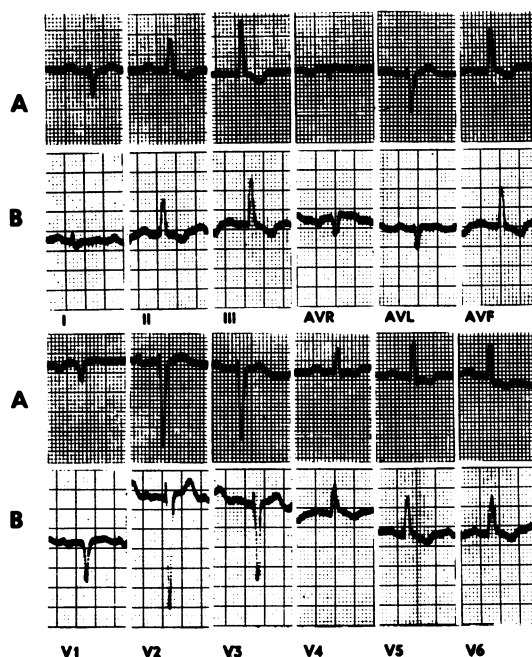


FIG. 3 Electrocardiogram (standard and augmented limb leads top and precordial leads bottom) showing left ventricular hypertrophy and left atrial enlargement. Tracing A shows temporary right axis deviation related to pulmonary embolism.

disease; they were more likely to be the result of digitalis. Excluding these cases it will be seen that conduction defects were observed almost twice as commonly in the Coloureds (34.6%) as compared to the Bantu (18.8%). By standard tests for differences of two proportions in large samples, the difference between the two groups was significant at the 1 per cent level ($P = 0.01$).

Comment

Conduction defects have been found to be more frequent in our cases of congestive cardiomyopathy

TABLE 2 Incidence of conduction abnormalities observed in 195 cases of congestive cardiomyopathy

Conduction defects	Coloured patients (78)		Bantu patients (117)	
	No.	Per cent	No.	Per cent
Left bundle-branch block	10	12.8	9	7.7
Left bundle-branch block and left axis deviation	5	6.4	0	
Left anterior hemiblock	12	15.4	13	11.1
Left anterior hemiblock and right bundle-branch block	0		0	
Right bundle-branch block	0		0	
First-degree AV block	3		7	
Total	30	(38.96)	29	(24.8)

than the reported incidence in hypertension, aortic valve disease, and among survivors of myocardial infarction, but less frequently and of different type to those reported in Chagasic cardiomyopathy (Pryor and Blount, 1966; Rosenbaum, 1964, 1969; Orndahl, Thelesius, and Hood, 1972; Rosenbaum, Elizari, and Lazzari, 1970). The tracings are similar to the latter condition, however, in that typical right ventricular hypertrophy was not observed and right axis deviation was rare and probably related to pulmonary embolism. These findings are related to predominant left ventricular dilatation resulting in normally directed electrical forces. The electrocardiograms thus present a fairly characteristic picture always showing left ventricular hypertrophy, frequently left atrial enlargement, absence of right ventricular hypertrophy, and right bundle-branch block, and rarely right axis deviation. Conduction defects occur in one-quarter of the cases, with left bundle-branch block and left atrial hypertrophy equally common.

The electrocardiographic findings in Coloureds with this disease have not been studied before and the only other findings in the South African Bantu available for comparison are those of Schamroth and Blumsohn who found that in 48 patients, 46 per cent had left anterior hemiblock and 8 per cent had left bundle-branch block; the presence of right bundle-branch block was not commented upon. Also, in a series of 28 cases Lewis, van der Horst, and Gotsman (1971) found left anterior hemiblock in 8 instances (25%) and left bundle-branch block in 2 (14%). The differences between these findings and ours may be related to the smaller samples in both of these studies.

A consideration of the pathology helps to explain the conduction defects described. Characteristically the heart is hypertrophied and dilated, and this usually involves all chambers. In their study of the pathology, Higginson, Isaacson, and Simson (1960) found intraluminal thrombus in 48 of their 80 specimens. The left ventricle was involved in 45 per cent and the right in 21 per cent of their cases. Organization of this thrombus which results from stasis leads to a mild degree of diffuse subendocardial fibrosis with the formation of avascular plaques. The underlying myocardium shows hypertrophied muscle fibres and occasional areas of intramyocardial fibrosis in relation to recently organized thrombus. The atrioventricular and semilunar valves are normal and the coronary arteries free of atherosclerosis.

Rosenbaum has shown that the main divisions of the left bundle-branches of the conducting system lie entirely in the subendocardium, whereas a portion of the longer right bundle lies within the myocardium (Rosenbaum, 1964). Recently Demoulin

and Kulbertus (1972) have shown in their histopathological studies of the hemiblocks that the left-sided Purkinje system is made up of three, rather than two, main networks which are widely interconnected and may have a complicated plexus of ramifications. These findings may explain the absence of right bundle-branch block in our cases and its frequency (30–60%) in Chagasic cardiomyopathy. During its intramyocardial course the right bundle is highly susceptible to the panmyocarditis of Chagas disease (Rosenbaum, 1964), whereas in congestive cardiomyopathy, only some of the peripheral Purkinje fibres would be involved, and the intramyocardial portion of the main right bundle is spared. The main subendocardial branches of the left bundle would obviously be susceptible to fibrosis which in any event is more common in the left ventricle (Becker, 1957).

We believe, therefore, that in our cases the left axis deviation and left bundle-branch block are a result of 'divisional' rather than 'predivisional' involvement of the left bundle. A frequent cause of predivisional left bundle-branch block is coronary artery disease, which may also play a role in the conduction defects found in hypertension and aortic valve disease (Rosenbaum, 1969). An ischaemic basis is extremely unlikely since the Bantu population is almost immune to coronary atheroma and the disease is uncommon in the Coloured (Seftel, Keeley, and Walker, 1963; Schrire, 1971). The examples of left bundle-branch block with the mean axis deviated to the left, which were encountered in our series, could be explained by complete anterior hemiblock and partial block in the posterior division, without invoking main stem left bundle-branch block with coexisting left anterior hemiblock (Desser and Benchimol, 1972).

The significant difference in the incidence of conduction defects in our patients suggests that the Coloured suffers from a more severe form of cardiomyopathy than the Bantu and it is tempting to speculate why this may be so. A study of the prognosis of this disease in the Bantu has shown that the majority die within 5 years (Keeley *et al.*, 1963). Unfortunately, the disease has received little attention in the Coloured and the prognosis is unknown.

The aetiology of myopathic heart failure in the Bantu of Johannesburg has been extensively reviewed recently by Seftel, Metz, and Lakier (1973). At least three factors appear to be operative: (1) thiamine deficiency, (2) excessive consumption of alcohol, and (3) chronic general malnutrition. Beriberi which has been recognized in Cape Town and Johannesburg is clearly related to thiamine deficiency and is directly conditioned by alcoholism (Grusin, 1957; Schrire and Gant, 1959). Cardio-

myopathy in children and the type occurring in the puerperium appear to be related to chronic general malnutrition, and heart failure may be precipitated by the nutritional drain of lactation or other stress (Seftel and Susser, 1961; Chesler *et al.*, 1971). A combination of factors appears to be operative in the largest group of cardiomyopathy cases. Seftel *et al.* (1973) have pointed out that the role of alcohol in the aetiology of the Bantu cardiomyopathy has received insufficient attention, and have shown that alcoholism is very common among the Johannesburg Bantu, particularly among men. Half of their patients with congestive cardiomyopathy, nearly twice the proportion found in the control group, were heavy drinkers, and alcohol was an important, if not the major, factor responsible for their heart disease; furthermore, remission may occur with abstinence.

There have been no comparable studies performed in Coloureds suffering from congestive cardiomyopathy, and we were unable to assess this feature accurately in our retrospective electrocardiographic study; it can be said, however, that the records noted a high incidence of heavy drinking in many of these patients. The alcoholic habits of the Coloured population as a whole, however, have been studied by Gillis, Lewis, and Slabbert (1965). The reports show that the frequency of chronic alcoholism is alarming. Allowing for slight differences in definition, Jellinek (1952) found that addictive and pre-addictive drinkers did not exceed 5 to 6 per cent of all users of alcohol in any country. In the survey for the Coloured population by Gillis *et al.* the figure is 18.2 per cent.

Chronic alcoholism may lead to cardiac damage and congestive cardiomyopathy (Burch and Giles, 1971; Evans, 1961). There are no gross or histological changes typical of alcoholic cardiomyopathy, but in the advanced stages of the disease the heart is enlarged with areas of fibrosis and endocardial thickening, findings similar to those reported in Bantu congestive cardiomyopathy. Electrocardiographic conduction defects are merely a reflection of this damage to the left ventricle, and our findings indicate that the Coloured patient suffers more severely than the Bantu; from circumstantial evidence it is tempting to postulate that alcohol may have a causative role. Further study is clearly needed to determine the incidence, prognosis, and role of alcohol in the genesis of congestive cardiomyopathy in the Coloured.

We wish to thank the Medical Superintendent of Groote Schuur Hospital, Dr. J. G. Burger, for permission to publish; and the South African Medical Research Council and the Cape Town City Council for financial support.

References

- Becker, B. J. P. (1957). Some African cardiomyopathies. *South African Medical Journal*, **31**, 854.
- Becker, B. J. P., Chatgidakis, C. B., and Van Lingen, B. (1953). Cardiovascular collagenosis with parietal endocardial thrombosis. *Circulation*, **7**, 345.
- Burch, G. E., and Giles, T. D. (1971). Alcoholic cardiomyopathy. *American Journal of Medicine*, **50**, 141.
- Chesler, E., Joffe, H. S., Beck, W., and Schrire, V. (1971). Tetralogy of Fallot and heart failure. *American Heart Journal*, **81**, 321.
- Demoulin, J. C., and Kulbertus, H. E. (1972). Histopathological examination of concept of left hemiblock. *British Heart Journal*, **34**, 807.
- Desser, K. B., and Benchimol, A. (1972). Transient complete left bundle branch block and left anterior hemiblock demonstrated by biplane timed vectorcardiograms. *Journal of Electrocardiology*, **5**, 90.
- Evans, W. (1961). Alcoholic cardiomyopathy. *American Heart Journal*, **61**, 556.
- Gillanders, A. D. (1951). Nutritional heart disease. *British Heart Journal*, **13**, 177.
- Gillis, L. S., Lewis, J. B., and Slabbert, M. (1965). *Psychiatric Disturbance and Alcoholism in the Coloured People of the Cape Peninsula*. University of Cape Town Press.
- Grusin, H. (1957). Acute reversible heart failure in Africans. *Circulation*, **16**, 27.
- Higginson, J., Isaacson, C., and Simson, I. (1960). The pathology of cryptogenic heart disease. *Archives of Pathology*, **70**, 497.
- Jellinek, E. M. (1952). Phases of alcoholic addiction. *Quarterly Journal of Studies in Alcoholism*, **13**, 673.
- Keeley, K. J., Chesler, E., Seftel, H. C., and Walker, A. R. P. (1963). Tropical cardiomyopathy. *Lancet*, **2**, 524.
- Lewis, B. S., Van der Horst, R. L., and Gotsman, M. S. (1971). Diagnostic electrocardiographic patterns in Bantu myocardiopathy and constrictive pericarditis. *South African Medical Journal*, **45**, 1110.
- Orndahl, G., Thelesius, O., and Hood B. (1972). Incidence of persistent atrial fibrillation and conduction defects in coronary heart disease. *American Heart Journal*, **84**, 120.
- Pryor, R., and Blount, S. G. (1966). The clinical significance of true left axis deviation. *American Heart Journal*, **72**, 391.
- Rosenbaum, M. B. (1964). Chagasic myocardiopathy. *Progress in Cardiovascular Diseases*, **7**, 199.
- Rosenbaum, M. B. (1969). Types of left bundle branch block and their clinical significance. *Journal of Electrocardiology*, **2**, 197.
- Rosenbaum, M. B., Elizari, M. V., and Lazzari, J. O. (1970). *The Hemiblocks: New Concepts of Intra-ventricular Conduction Based on Human Anatomical, Physiological and Clinical Studies*. Tampa Tracings, Oldsmar, Florida.
- Schamroth, L., and Blumsohn, D. (1961). The significance of left axis deviation in heart disease of the African. *British Heart Journal*, **23**, 405.
- Schrire, V. (1964). The racial incidence of the less common forms of heart disease at Groote Schuur Hospital 1952-1961. *South African Medical Journal*, **38**, 598.
- Schrire, V. (1971). Heart disease in Southern Africa with special reference to ischaemic heart disease. *South African Medical Journal*, **45**, 634.
- Schrire, V., and Gant, J. (1959). The electrocardiographic changes associated with beri-beri heart disease. An analysis of 50 cases studied at Groote Schuur Hospital, Cape Town during a period of five years. *South African Journal of Laboratory and Clinical Medicine*, **5**, 195.

- Seftel, H. C., Keeley, K. J., and Walker, A. R. P. (1963). Characteristics of South African Bantu who have suffered from myocardial infarction. *American Journal of Cardiology*, **12**, 148.
- Seftel, H. C., Metz, J., and Lakier, J. B. (1973). Aetiology of cardiomyopathy in Johannesburg Bantu. *South African Medical Journal*, **47**, 273.
- Seftel, H. C., and Susser, M. (1961). Maternity and myocardial failure in African women. *British Heart Journal*, **23**, 43.
- Requests for reprints to Dr. E. Chesler, Department of Medicine, University of Cape Town, Cape Town, South Africa.